

Study of Long-Latent Disease In Industrial Populations

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We all recognize that our decisions concerning the control of environmental pollutants must frequently be based on incomplete information and imperfect measurement of exposure to toxic agents and of disease response. This is especially true in the case of diseases that appear many years after exposure to the toxic agent. Because lack of sufficiently detailed information may lead to misinterpretation regarding causal relationships, it is important to consider some of the pitfalls in the study of these diseases.

The material I am presenting here was prepared primarily to demonstrate how we identify occupational groups at excess risk of long-latent disease and how evidence is developed on cause-effect relationships. At the same time, because I am particularly concerned with the possible misinterpretation of epidemiological findings, I have tried to emphasize some basic principles that are frequently ignored and which may lead to erroneous conclusions of a cause and effect relationship or the lack of a cause and effect relationship.

The first point I should like to make is that in studying the relationship between occupational exposure and disease that appears many years after exposure, we are immediately limited as to the population groups to be studied, the way in which exposure can be characterized, and the disease entities to be studied. Thus, the study of currently employed industrial populations would be inappropriate for identifying effects related to exposures many years in the past, unless the turnover of the work force

was extremely low. The obvious answer to this problem is to identify populations that have been employed in specific occupations many years in the past and to undertake a prospective study in retrospect of their disease experience. This we have been able to do in a number of industrial situations, including the one which I shall describe shortly. Unfortunately, such an approach imposes severe limitations on our measurement of disease response. For most industrial populations, information on the early stages of disease is available only for the currently employed survivors. Consequently, our studies of long-term disease are usually restricted to an analysis of mortality patterns, and as a further consequence must be limited to the fatal diseases in which we might expect that mortality provides a good index of disease incidence.

The population chosen for study consisted of 59,000 steelworkers employed at 7 plants of 3 major steel firms in Allegheny County, Pennsylvania in 1953. Employment records on these men were recovered in 1962 and follow-up was initiated to determine vital status through 1961. As shown in Fig. 1, only 32,263 of the original study population had continued employment into 1962. It is thus seen that a study of current employees to determine the relationship between health status and prior exposures of the study cohort would exclude almost one half of the exposed population.

Thirteen percent of the study cohort had retired during the observation

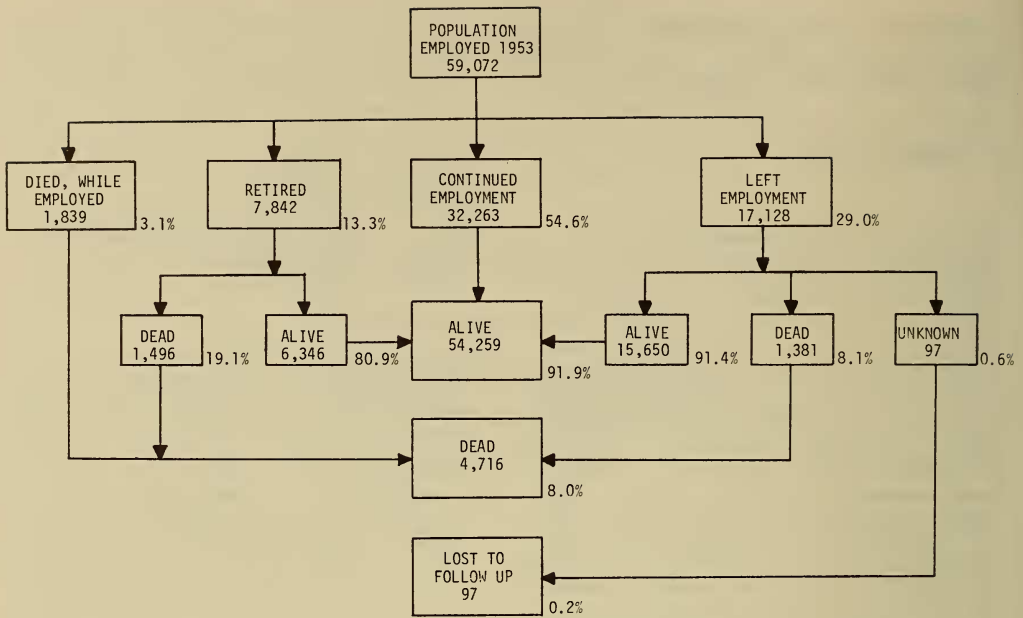


Fig. 1.—Followup of Allegheny County steelworkers population showing initial employment status and vital status at end of study.

period and the 1,500 deaths within this group could be identified by reference to company records. Another 3% of employees had died between 1953 and 1962 while employed. A frequently used approach to the study of industrial mortality is to relate these deaths to the average employment level during the period of study or to contrast the proportionate mortality due to specific causes among retirees with some standard group. Again, it should be noted that if we are concerned with relating disease response to prior exposures, we would be ignoring the experience of the 17,000 men who had left employment, and whose vital status could not be determined by reference to employer records. Extensive follow-up of those who had left employment showed that 8% of this group had expired before 1962, about the same level of mortality as seen for the total cohort. However, when we recognize that the population and deaths which could be followed through reference to employer records are heavily loaded with retirees in the older ages, it is seen that the total mortality rate for those who left

employment is actually higher than for those who remained. As seen in the box at the bottom of Fig. 1, vital status was eventually determined for all but 97, or 0.2%, of the original study cohort.

A further consideration in deciding whether records available to the employer might be appropriate for the study of work related mortality, is whether these records reflect the same distribution of cause-specific mortality as would be seen in the original cohort. The figures displayed in Table 1 further emphasize the limitation of studies based on these records. Here it is seen that there is considerable variation in the percentage of deaths identifiable by employer records according to specific cause of death. As might be expected a large part of deaths from diseases of the nervous system and the circulatory system can be identified by employer records because of the high mortality from heart disease and from strokes among the retirees and men with long service. On the other hand, almost 45% of deaths due to malignant neoplasms were unknown to the employer. Even within the malignant neoplasms there is con-

Table 1.—Number of deaths from selected causes by employment status at death and knowledge of employer (Allegheny County steelworkers, 1953–1961).

Cause of Death	International List Number	Total Deaths	Known to Employer		Unknown To Employer	Percent Unknown
			At Work	Retired		
All Causes	(001-999)	4,716	1,839	1,496	1,381	29.3
Malignant neoplasms, all sites	(140-205)	1,008	212	346	450	44.6
Lung and Bronchus	(162-163)	295	68	87	140	47.5
Prostate	(177)	56	7	36	13	23.2
Brain and other nervous system	(193)	28	3	3	22	78.6
Diseases of nervous system and sense organs	(330-398)	382	136	174	72	18.8
Diseases of circulatory system	(400-468)	2,001	905	672	424	21.2
Arteriosclerotic heart disease, including coronary	(420-422)	1,680	792	543	345	20.5
Diseases of respiratory system	(470-527)	165	57	55	53	32.1
Diseases of digestive system	(530-587)	267	109	74	84	31.5
Accidents	(800-965)	356	208	39	109	30.6

siderable variation in the percentage of deaths unknown to the employer, ranging from 23% for cancer of the prostate, a group which is heavily loaded with retirees, to 78.6% of deaths from malignancies of the brain and central nervous system. It is thus seen that the mortality studies based on employer records must be interpreted with caution unless we can be assured of an extremely low turnover of personnel.

The next important factor to consider in the study of occupational disease is the choice of a control or comparison group. I think you would find general agreement in the field of occupational studies that there is no such thing as the "ideal" control population with which to compare our industrial populations. Usually it is a question of which of several comparison groups is the least biased, and in many cases the only available data for contrast is that for the general population. As a consequence, we continue to see reports in the occupational health literature of unusually low mortality for certain industrial groups contrasted with the general population, with the implication that no health hazards exist. However, when we consider that for many industrial

populations a certain level of good health is required for employment and continuing good health is required for continued employment, this is exactly the pattern we would expect to see. As shown in Table 2 this is what we did observe for the steelworker population in contrast with the general population of Allegheny County. From 1953 through 1961 we observed 4,716 deaths in steelworkers where we would have expected to see 5,767. The same pattern is observed for each of the specific causes of death with the exception of accidents. This is not surprising, since we would expect more accidents in an industrial population. The extent to which such comparisons might lead to erroneous conclusions is also seen to vary both by cause and by race. For example, mortality from the infectious and parasitic diseases is less than 40% of expectation, whereas mortality from the malignant neoplasms in steelworkers is about 90% of expectation. It is also seen that the deficit in mortality is considerably greater for the non-white workers than for white, mortality for the former group being only 64% of expectation. Particularly striking is the 53% deficit for vascular lesions affecting the

Table 2.—Observed and expected deaths from selected causes by race (Allegheny County Steelworkers, 1953–1961).

Cause of Death	List Number	Total		White		Nonwhite	
		Observed	Expected	Observed	Expected	Observed	Expected
All Causes		4716	5766.8	4083	4773.5	633	993.3
Infective and parasitic diseases	001-138	65	166.9	39	94.7	26	72.2
Malignant neoplasms	140-205	1008	1091.4	861	929.0	147	162.4
Vascular lesions affecting C.N.S.	330-334	365	464.3	310	359.8	55	104.5
Heart disease	400-443	1906	2311.3	1721	2012.5	185	298.8
Arteriosclerotic and deg. heart disease	420-422	1680	2002.9	1537	1773.4	143	229.5
Other heart disease	(400-416) (430-443)	226	308.4	184	239.1	42	69.3
Diseases of respiratory system	470-527	165	237.6	136	178.4	29	59.2
Accidents	800-962	356	311.8	302	252.6	54	59.2
Homicide and suicide	(963-964) (970-985)	118	138.7	94	100.7	24	38.0
All other causes	Residual	733	1044.8	620	845.8	113	199.0

*Expected deaths calculated by applying age, race and calendar year specific rates of the study county to steelworker person years at risk.

central nervous system observed for non-white steelworkers.

For study of specific occupational groups within the steel industry, we have used the total steelworker population as the control group. This is, of course, preferable to comparison with the general population in that it overcomes selection related to employability. At the same time, it should be recognized that other selective factors may be operating. Principal among these is selection for health which may be expected to occur within specific work areas. Thus for certain occupations that are very demanding and require good health for initial and continued employment, the less healthy individual would be expected to choose work in other areas or, by company policy, be assigned to less demanding areas when ill health develops. Consequently, certain work areas may show marked deficits or excesses in mortality unrelated to exposure in the occupational environment.

Another important and frequently overlooked factor to be considered is that striking differences in mortality may be expected by chance alone when we make many comparisons. This is seen in Table 3 which displays some of the comparisons for men employed in

1953 in each of 53 work areas within the steel industry. Although such a division is not unexpected, it should be noted that almost half of the work areas show a lower mortality rate than expected (28 above and 25 below). Because of concern with potential hazards in the occupational environment, many studies of occupationally related disease have been directed at the identification of employed groups showing excess disease, and deficits have been mostly ignored. However, deficits in mortality may indicate the selection of more healthy individuals into certain work areas and, as a possible consequence, the assignment of less healthy individuals to other areas. For that reason, an evaluation of the relationship between the area of employment and consequent mortality must include an assessment of both excesses and deficits. We note in Table 3 that significant excesses in total mortality were seen for janitors and men working in the machine shop, while a significant deficit in mortality is noted for men working in the carpenter shop. The excess for janitors was predictable, and is not related to occupational exposures. Rather, the high mortality for this group is due to assignment of workers from other areas because of ill health. We should also note in Table 3 that the

Table 3.—Number employed in 1953, observed and expected deaths from all causes and standardized mortality ratios by work area (Allegheny County steelworkers, 1953–1961).

Work Area	Number Employed	Observed Deaths	Expected Deaths	SMR ¹
All steelworkers	58,828	4,685	4,685.0	100
Annealing normalizing	1,367	121	109.2	111
Batch pickl. - sheet dry.	76	6	5.8	103
Billet bloom & slab	3,136	290	277.5	105
Blacksmith shop	271	26	28.2	92
Blast furnace	3,455	244	265.6	92
Carpenter shop	406	29	40.9	71*
Coating	332	19	23.4	81
<u>Coke plant</u>	<u>2,552</u>	<u>206</u>	<u>198.9</u>	<u>104</u>
Cont. pickl. & elec. clean.	205	12	9.8	122
Cold reducing mills	1,499	100	87.1	115
Electric main. assg.	1,616	118	127.3	93
Electric furnace	742	46	43.8	105
Electric shop	561	39	49.5	79
Foundry	1,143	90	101.1	89
Gen. admin. & clerical	3,312	224	240.8	93
Gen. finish & ship.	473	38	40.2	95
Gen. receiv. & stores	196	17	19.0	89
Gen. technical	2,373	123	130.1	95
General labor	969	84	78.4	107
Heat treat. & forging	1,015	75	78.1	96
Hot pack mills	572	55	66.6	83
Hot strip mill	161	19	17.0	112
Hot strip rolling	1,014	63	73.9	85
Janitors	521	113	84.5	134**
Loco & car repair	129	11	11.7	94
Machine shop	1,450	151	130.2	116*

$$^1\text{SMR} = \frac{\text{Observed deaths}}{\text{Expected deaths}} \times 100$$

Significance based on summary chi-square with 1 degree of freedom.

* 5% level

** 1% level

mortality for coke plant workers, which will be discussed in detail, appears to be little different from that of other steelworkers.

One of the most serious deficiencies in the study of long-term disease is that we only rarely have access to measurement data which depict the extent of exposure to specific toxic agents in the work place. In the present study, for example, degree of exposure could only

be characterized in terms of length of employment within specified work areas and in a few cases by reference to recent measurements within smaller sub-divisions in these work areas. As crude as these measures are, we would expect that they would reflect any dose-response relationship unless there was a rapid turnover of the work force due to early removal of "more sensitive" individuals. An example is shown

in Table 4 where we have noted the work areas which show a significant excess or deficit in total mortality for persons employed five or more years in each work area. Here it is seen that significant differences in mortality have been noted for several of the work areas which were not noted in Table 3. An interesting observation, for which figures are not shown in Table 3, is the 25% excess in total mortality for white steelworkers employed as general laborers. White steelworkers employed in this area in 1953 showed a slight deficit in mortality during the study period. This suggests that the inclusion of many short-term employees may have masked important differences. We also notice in Table 4 that the total mortality experience of white and non-white workers employed at the coke plant for 5 years is quite different. While the non-whites show a 22% excess in total mortality, the experience for the white workers is about as expected.

Another problem in limiting our analysis to persons employed in a single year, such as 1953, is that persons

suffering ill health may migrate to other work areas. Consequently, we may draw erroneous conclusions about health status from our observation of the more healthy survivors. To illustrate how extensive this problem might be, we show in Table 5 the distribution of workers employed in the 2 major subdivisions of the coke plant in 1953 and in prior years. This subdivision into coke oven and non-oven areas was based on previous information which suggested that excess mortality of coke plant workers might be associated with exposure to coke oven effluent. Of the 59,000 steelworkers employed in 1953, 2,552 were working in the coke plant. However, an additional 978 men employed in other areas in 1953 had been employed at the coke plant in some prior year. Thus, limiting the study to those employed in 1953 would exclude 28% of men with prior coke plant exposure. It is also seen that the distribution of white and non-white workers is considerably different for oven and non-oven areas, and that the proportion of coke oven workers

Table 4.—Persons years at risk, observed and expected deaths from all causes and standardized mortality ratios for men employed at least 5 years in specified work areas (Allegheny County steelworkers, 1953–1961).

Work Area	Race	Person Years at Risk	Observed Deaths	Expected Deaths	SMR
Batch pickling-sheet drying	Total	525	18	9.9	182*
Carpenter	Total	2,963	30	44.4	68*
Coke Plant	White	11,549	130	130.8	99
	Nonwhite	6,509	96	78.7	122*
Cold reducing mills	White	8,204	71	70.7	100
	Nonwhite	593	14	6.3	222**
General finish. and ship.	White	4,385	69	64.4	107
	Nonwhite	1,041	8	16.9	47*
General labor	White	4,989	102	81.6	125*
	Nonwhite	3,636	49	46.1	106
Janitors	White	1,117	54	34.5	157**
	Nonwhite	2,015	33	33.1	100
Maintenance NOS	Total	2,246	10	28.9	35**
Mech. main. assg.	White	22,313	377	318.4	118**
	Nonwhite	721	16	14.3	112
Merchant mills	White	18,372	253	266.9	95
	Nonwhite	2,966	28	42.4	66*
Sheet fin. and ship.	White	13,466	158	133.5	118*
	Nonwhite	438	5	7.3	68

* 5% level

** 1% level

Table 5.—Distribution of Allegheny County Coke plant workers by work area, race, and period of employment.

	Coke Plant	Coke Oven		Nonoven	
		Number	Percent	Number	Percent
<u>Employed in 1953</u>					
Total	2,552	1,327	52.0	1,225	48.0
White	1,645	520	31.6	1,125	68.4
Nonwhite	907	807	89.0	100	11.0
<u>Employed in 1953 or Prior Years</u>					
Total	3,530	2,048	58.0	1,482	42.0
White	2,369	993	41.9	1,376	58.1
Nonwhite	1,161	1,055	90.9	106	9.1

excluded by an analysis limited to 1953 is greater for whites than for non-whites. Overall, 35% of men with prior coke oven exposure would not be included in such an analysis, while 42% of white coke oven workers would be excluded.

Mortality from specific causes for the coke oven workers is shown in Table 6. Here it is seen that of the 184 deaths among men previously employed at the coke ovens, only 100 deaths would have

been related to this area in 1953. As regards the cause-specific mortality of these workers, it is seen that the greatest part of the excess mortality for the non-white coke oven workers is due to a significant excess in respiratory cancers. While the differences are not significant, the respiratory cancer mortality for white coke oven workers also suggests the possibility of an excess risk for this disease. Looking now at Table 7, we see that the only suggestion of an

Table 6.—Observed and expected deaths and standardized mortality ratios for men employed in 1953 and in prior years (Allegheny County coke oven workers, 1953-1961).

Cause of Death	Employed in 1953			Employed in 1953 or Prior		
	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
<u>White</u>						
All Causes	25	31.4	80	80	80.2	100
Malignant neoplasms-respiratory system	3	1.9	-	8	5.0	160
Malignant neoplasms-digestive organs and peritoneum	1	2.4	-	6	6.1	98
Other malignant neoplasms	2	2.1	-	6	5.8	103
Vascular lesions affecting CNS	1	2.3	-	6	6.4	94
Heart disease	9	12.8	70	29	32.9	86
Diseases of respiratory system	0	1.0	-	2	2.5	-
All other causes	9	8.9	101	23	20.5	112
<u>Nonwhite</u>						
All Causes	75	65.2	115	104	92.9	112
Malignant neoplasms-respiratory system	17	5.7	298**	25	8.4	298**
Malignant neoplasms-digestive organs and peritoneum	3	4.1	-	3	5.7	53
Other malignant neoplasms	8	5.6	143	10	8.3	120
Vascular lesions affecting CNS	6	5.6	107	9	7.8	115
Heart disease	13	18.9	69	20	27.6	72
Diseases of respiratory system	4	3.0	-	5	4.2	119
All other causes	24	22.4	107	32	31.0	103

Table 7.—Observed and expected deaths and standardized mortality ratios for men employed in 1953 and in prior years (Allegheny County non-oven workers, 1953–1961).

Cause of Death	Employed in 1953			Employed in 1953 or Prior		
	Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
	<u>White</u>					
All Causes	89	89.7	99	119	113.6	105
Malignant neoplasms-respiratory system	1	5.7	18	3	7.3	41
Malignant neoplasms-digestive organs and peritoneum	10	7.4	135	14	8.8	159
Other malignant neoplasms	5	6.0	83	4	8.2	49
Vascular lesions affecting CNS	12	7.2	167	11	8.9	124
Heart disease	38	37.4	102	58	47.6	122
Diseases of respiratory system	6	3.1	194	5	3.9	128
All other causes	17	23.0	74	24	28.9	83
	<u>Nonwhite</u>					
All Causes	17	12.6	135	17	13.8	123
Malignant neoplasms-respiratory system	4	1.1	-	1	1.2	-
Malignant neoplasms-digestive organs and peritoneum	2	0.8	-	3	0.9	-
Other malignant neoplasms	3	1.2	-	2	1.4	-
Vascular lesions affecting CNS	0	1.1	-	2	1.2	-
Heart disease	4	3.9	-	4	4.1	-
Diseases of respiratory system	2	0.6	-	2	0.6	-
All other causes	2	3.8	-	3	4.4	-

excess for respiratory cancer was in the non-whites who worked in the non-oven area in 1953. Since men employed only in the non-oven area in 1953 or prior years show a deficit for respiratory cancer, this would suggest that these deaths are associated with exposure at the coke ovens in years prior to 1953. Analysis of the data with reference to place of employment in 1953, therefore, would have attributed these deaths to exposure in the non-oven area.

Further subdivision of the coke oven population according to exposure levels, as defined by work assignments and years of employment, demonstrates the relationship between exposure to coke oven effluent and lung cancer response. This is shown in Table 8. Here we see that the excess of lung cancer is associated with 5 or more years employment at the coke ovens and that the level of risk increases with amount of exposure, the greatest exposure to effluent being at the top side of the ovens. These findings also serve to illustrate how occupationally-related disease might be masked by limiting study to broad occupational groups.

Since the top-side workers constitute only 15% of coke oven workers and only 9% of coke plant workers, the extremely high risk for top-side workers is reflected as a considerably lower relative risk for coke oven workers and coke-plant workers. If the lung cancer rate for top-side workers had been only double the rate for other steel workers, we would have failed to note a significant excess for coke oven workers, while a 5-fold risk would have been insufficient to demonstrate a significant excess for coke-plant workers. A similar diluting effect may result from inclusion in the study group of coke oven workers with too few years of observation to allow for the appearance of latent effects.

Finally, it should be pointed out that before we draw inferences about causation, we should, to the extent that information is available, rule out other factors which might explain the relationship. While we could not identify any artifactual relationship which would produce such a unique picture only for Allegheny County, it was suggested that replication of this study in other geo-

Table 8.—Number employed, observed and expected deaths and standardized mortality ratios for selected causes by length of employment and work area in 1953 and prior years (Allegheny County coke oven workers, 1953–1961).

Work Area and Length of Employment	Number Employed	All Causes			Malignant Neoplasm of Lung		
		Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
Coke Oven, < 5 Years	1,144	67	72.8	92	4	4.7	-
Coke Oven, ≥ 5 Years	904	117	100.3	117	27	7.6	355**
Side Oven Only	496	53	55.1	96	6	4.1	146
Side and Topside	276	29	27.9	104	6	2.1	286**
Topside	132	35	17.4	201**	15	1.5	1,000**
		Other Malignant Neoplasms			Diseases of Resp. System		
		Observed Deaths	Expected Deaths	SMR	Observed Deaths	Expected Deaths	SMR
Coke Oven, < 5 Years		7	10.5	67	0	2.8	-
Coke Oven, ≥ 5 Years		20	16.4	122	7	4.0	175
Side Oven Only		7	8.9	79	3	2.3	-
Side and Topside		9	4.3	209*	3	1.0	-
Topside		4	3.2	-	1	0.7	-

graphical areas would provide additional evidence on the subject. Further study of coke oven workers throughout the United States and Canada was undertaken and some of the findings are shown in Table 9. Here it is seen that coke-oven workers from other geographical areas

also experienced unusually high lung cancer mortality; that the highest risk is observed for the top-side workers; and that men employed at the coke ovens also are at excess risk of genito-urinary cancer. A second point to be considered in determining causality is whether the

Table 9.—Observed and expected deaths, 1951–1966, and relative risk for selected causes for coke oven workers employed during 1951–1955 at 10 non-Allegheny County plants for coke oven workers employed during 1953 at 2 Allegheny County steel plants.

Cause of Death	Full Topside			Partial Topside			Side Oven		
	Obs. Deaths	Exp. Deaths	Rel. Risk	Obs. Deaths	Exp. Deaths	Rel. Risk	Obs. Deaths	Exp. Deaths	Rel. Risk
All Causes	157	144.1	1.12	73	69.2	1.07	359	379.7	0.92
Malignant neoplasms—lung, bronchus, and trachea	35	10.4	7.24**	7	3.7	2.14	27	19.4	1.73*
Malignant neoplasms—genito-urinary organs	4	2.5	-	2	0.8	-	15	9.2	2.02*
Other malignant neoplasms	15	16.8	0.86	5	9.7	0.44	35	41.4	0.79
Tuberculosis of the respiratory system	3	2.3	-	0	0.3	-	5	4.8	1.06
Other diseases of the respiratory system	7	7.0	0.99	5	3.1	1.78	18	17.8	1.01
Cardiovascular renal diseases	60	68.5	0.84	39	33.8	1.18	155	180.9	0.80*
Accidents	10	13.4	0.72	7	7.4	0.94	45	40.1	1.19
All other causes	23	23.1	1.00	8	10.2	0.76	59	66.2	0.85

Significance of Relative Risk (Rel. Risk) based on summary chi-square with one degree of freedom.

* 5% level

** 1% level

- less than five deaths

findings are consistent with other comparable observations. A review of the literature shows that all of the occupational groups engaged in the carbonization of bituminous coal or in handling of the by-products are at excess risk of cancer for 1 or more sites. More specifically, all of the 3 populations engaged in the carbonization of bituminous coal have shown a striking excess of lung cancer and the lung cancer response is positively associated with the temperature of carbonization.

One final factor which must be considered, because of the recognized association with lung cancer mortality, is the possible role of cigarette smoking in the unusual mortality experience of the coke oven workers. Unfortunately, as with most long-term studies, no smoking histories are available. However, it is possible to determine by reference to lung cancer mortality rates for cigarette

smokers in the United States whether the unusual lung cancer experience of the coke oven workers might be explained by differences in smoking habits. As seen in Table 10, while the total steelworker population shows a lung cancer mortality somewhat like that observed for all cigarette smokers, and the coke-oven workers, never topside, show rates not too different from those for heavy cigarette smokers, the rates for top-side workers and for those employed more than 5 years top-side are far beyond what would have been predicted by differential cigarette smoking experience. While the possibility of a synergistic relationship between cigarette smoking and exposure to coke oven effluent certainly cannot be ruled out, we can say that the unusual lung cancer mortality experience of the coke oven workers cannot be accounted for by cigarette smoking habits alone.

Table 10.—Estimates of average annual lung cancer mortality rates (per 100,000 person-years) for selected U.S. smoking groups, 1954–1962 and steelworker groups, 1953–1961.

A G E	U.S. Smokers	35-44	45-54	55-64	65-74
	Steelworkers	<45	45-54	≥55	
Never smoked or occasional only	-	-	-	12	29
Current cigarette smokers - total	5	39	-	158	258
Current cigarette smokers, 1-9/day	-	-	-	69	119
Current cigarette smokers, over 39/day	-	-	104	321	559
Steelworkers	-	12	126	-	160
Coke oven, never topside	-	-	-	130	387
Coke oven, topside	-	228	1,058	-	1,307
Coke oven, ≥5 years topside	-	265	1,587	-	1,961